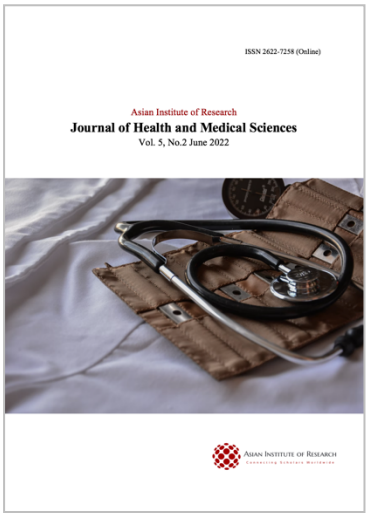

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Perioperative Anaesthesia Management for Aorta Dissection Patient Undergo Bentall Procedure

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Abstract
Introduction: aortic dissections that involve the ascending aorta (Stanford type A) are considered surgical emergencies. The mortality rate without emergency surgery is about 1% per hour for the first 48 hour, 60% by about one week, 74% by 2 weeks and 91% by 6 month. Open chest surgery with resection of dissected aorta may reduce the expected fatal outcomes to 10% as soon as the treatment provided in the first 24 hour and 20% for next 14 day. Case: A case of 42 years old man, 72 kg weight complained of sharp, tearing, pain from upper abdomen to chest and back of body since a month before admission. The pain endured for 20 minutes, patient had history of hypertension a year ago. Suspected with aortic dissection patient transferred to CVCU got therapy of antihypertension and β-blocker. Laboratory examination showed kidney disorder with enhancement of Ureum and creatinine levels. CT contras showed aortic dissection Stanford A Debakey Type I and patient scheduled for Bentall Procedure with complication Acute Kidney Injury (AKI). Perioperative anaesthesia management from preoperative with β-blocker and ramipril, induction with high dose analgetic, smooth intubation prevent increase in systolic blood pressure and heart rate, also maintain oxygen delivery to brain when DHCA started use SACP monitored with NIRS. after operation patient treated in ICU with ventilator and full sedated. Second day in ICU patient developed high creatinine levels and low urine output per hour. Renal replacement therapy is given and patient transfer to CVCU on the fifth day. Conclusion: it is still challenging to treatment of aortic dissection, started from diagnosis, preoperative management and overcoming the complication. Therefore, careful history taking, early treatment to prevented expansion of dissected aorta, CT angiography for diagnosed, intraoperative management and haemodialysis therapy should be considered to increase patient outcome.

Keywords: Perioperative Management, Aorta Dissection, Bentall Procedure, DHCA, AKI

1. Introduction

Aortic dissections that involve the ascending aorta (Stanford type A) are considered surgical emergencies. The mortality rate without surgery is about 1% for every first 48 hours, 60% by about 1 week, 74% by 2 weeks and 91% by 6 month (Kaplan,2017). Open chest surgery with resection of the dissected aorta may reduce the expected fatal outcome to 10% in the first 24 hour and 20% for the next 14 days. Therefore, operative management of

ATAAD (Acute type A aortic dissection) it's still accepted as gold standard for the management of this perilous condition (Petrov, 2020).

Aortic dissection results from an intimal tear that exposes the media to the pulsatile force of blood within the aortic lumen. Blood may exit the true aortic lumen and dissect the aortic wall to create a false lumen. The aortic dissection may remain localized at the primary entry site at the original intimal tear, or it may extend proximally, distally, or both (Kaplan,2017). The aetiology is frequently unknown but is related to hypertension (72%), and atherosclerosis (30%). Other predisposing factors are Marfan's syndrome, Ehlers-Danlos syndrome, coarctation, bicuspid aortic valve, Turner syndrome, aortitis, cocaine abuser, pregnancy, and blunt chest trauma (Dewi, 2020). The prevalence of AD has been reported approximately 5200/100.000 in western countries, with a male predominance of 3:1 and in approximately tw0-thirds of case involving the ascending aorta (Claussen, 2008). The incidence of Acute Aortic Syndrome (AAS) ranges from 3,5 to 6.0 per 100.000 patient-years in general population but increases in patients aged 64 to 74 years (27 per 100.000) and older than 75 years (35 per 100.000). acute aortic dissection comprises 85% to 95% of all AAS (Mussa, 2016).

Acute aortic dissection is defined as dissection occurring within 2 weeks of onset of pain. Subacute and chronic dissections occur between 2 and 6 weeks, and more than 6 weeks from the onset of pain respectively. Sudden onset of severe chest and/or back pain is the most typical symptom (Fukui, 2018). Extension of the dissection can lead to involvement of side branch vessels, resulting in cerebral, coronary, mesenteric, renal and limb ischemia (Pawan, 2008). Post-operative acute kidney injury (AKI) is a common complication with a high odds ratio 3.49 for 30-days mortality after operation (Hsiang,2021).

2. Case Report

A 42-year-old, 74 kg and height 180 cm complained of sharp, tearing, pain from upper abdomen to chest and back of body since a month before admission. The pain endures for about 20 minutes. Patient also complained shortness of breath especially during strenuous activities. Patient has history of hypertension since a year ago and patient received antihypertension drugs. Other history of disease such as stroke, asthma and Diabetes were declined. Patient treated in Cardiovascular Care Unit (CVCU) and has been given therapy. When examined, the patient appears calm, compos mentis, and don't feel pain. Blood pressure that time 199/57 mmHg with heart rate 56 bpm and respiratory rate 24 times per minute. Oxygen saturation 97% with room air. Chest physical examination revealed a diastolic murmur. The results of laboratory examinations showed that the values of hemoglobin, hematocrit, leukocytes and platelets were within normal limits. Electrolyte results showed hyponatremia (129 mg/dl) and hyperkalemia (5.1 mg/dl). Kidney function is also impaired with the value of urea (108 g/dl) and creatinine (2.24 g/dl). X-ray examination revealed cardiomegaly with interstitial lung edema, ECG examination gave sinus rhythm and LVH results. Another examination is CT Angiography with the conclusion of aortic dissection that extends to the brachiocephalic to the bifurcation common iliac artery along lk. 494 mm Stanford A Debakey I classification. TEE was also performed with the conclusion of mild regurgitation of the mitral, tricuspid, and pulmonary valves with normal right ventricular systolic function, widening of the aortic root and visible aortic dissection.

Table 1: Preoperative Laboratory

Hemoglobin	Hematokrit	Leukosit	Trombosit	SGOT	SGPT
15 g/dL	44,7 %	6330/mm^3	192.000/mm^3	37 U/L	42 U/L
Ureum	Kreatinin	Natrium	Kalium	Klorida	Magnesium
108 mg/dL	2,24 mg/dL	129 mEq/L	5,1 mEq/L	105 mEq/L	2,1 mg/dL

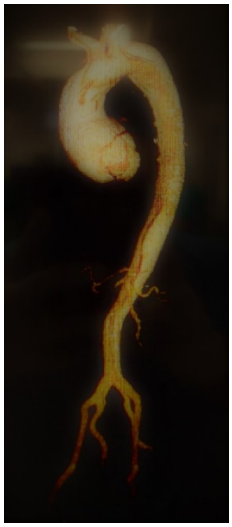

Figure 1: Three-dimensional results of CT Angiography showing Stanford A Debackey Type 1 aortic dissection.

The patient has been admitted to the CVCU for about 1 week and has been given oral therapy with ramipril 7.5 mg once a day, bisoprolol 10 mg once a day, ivabradine 5 mg twice a day, lansoprazole 30 mg once a day, and sucralfate syrup 10 cc three times a day.

3. Anesthesia Management

the patient's hypertension was controlled with oral therapy in the form of ramipril, bisoprolol, and ivabradine which were given while in CVCU. The patient is entered into the preparation room for an EKG for monitoring and an EKG for IABP (Intra-Aortic Balloon Pump), external defibrillator, IV line in the right hand, and arterial line in the left radial artery for invasive monitoring. the patient enters the operating room, monitoring equipment is connected including NIRS (Near Infrared Spectroscopy) which is installed in the frontal head of the patient's left and right, at that time the monitor shows blood pressure 110/69 mmHg, pulse rate 50 beats per minute, respiratory rate 18 breaths per minute and saturation 100%. The NIRS value when the patient is awake is the reference value for the patient, which is 64% for the right and left hemispheres.

After all monitoring devices were installed and working properly, induction was started by titrating midazolam 3 mg, fentanyl 225 mcg, propofol 50 mg and rocuronium 80 mg. Intubation was carried out after 3 minutes with a smooth ETT No. 8.0, depth of 21 cm, the anesthetic agent was turned on sevoflurane 1% and the ventilator control volume setting was 475 ml, peep 5 cmH2O, respiratory rate 12 times per minute and O2 fraction at 50%. Hemodynamic post intubation blood pressure 127/49 mmHg, pulse rate 52 beats per minute, respiratory rate 12 x per minute with 100% O2 saturation.

The operation was continued with CVC placement in the right subclavian vein, side port in the right internal jugular vein, and the second line artery in the left femoral artery. Phlebotomy as much as 2 bags of whole blood as much as 250 ml each. Insertion of Transesophageal Echocardiography (TEE) to see the morphology of the patient's heart and nasopharyngeal temperature. Fentanyl 100 mcg is injected five minutes before the incision. The incision was hemodynamically stable, five minutes after the incision heparin 300 mg was injected with an ACT value of 130 s and an ACT after heparinization of 597 s. Cardiopulmonary Bypass (CPB) was initiated 15 minutes post-incision with aortic cannulation of the right femoral artery and venous cannulation of the right atrium leading to the IVC. Deep Hypothermic Circulatory Arrest (DHCA) begins 118 minutes after CPB is working or 105 minutes after AOX, when DHCA is started CPB is stopped, while joining the prosthesis distal and proximal to the aorta at 25^{O}C and to avoid injury to the head being covered with ice. Anterograde Selective Cerebral Perfusion (ASCP) is used when DHCA is started to provide O2 supply to the left and right brain so that the NIRS value does not fall to 20% of the reference value. DHCA is finished when suturing is complete and CPB returns to work, the patient is warmed slowly to a temperature of 36^{O}C and evaluates the results of the operation.

Post CPB hemodynamically stable with blood pressure 103/68 mmHg to 108/71 mmHg, pulse rate 49 to 52 times per minute, respiratory rate 12 times per minute with cvp 8-12 cmh2O, and PAP 29/22 mmHg. The patient was given an inotropic at that time, dobutamine 10 mcg/kg/min, norepinephrine 0.1 mcg/kg/minute, followed by protamine 400 mg. The bradycardia was treated with the installation of an intracardiac pacemaker with the pulse rate set at 70 beats per minute. Total bleeding during surgery is estimated at 1100 cc. The operation lasted for 6 hours with stable hemodynamics including blood pressure ranging from 105/56 mmHg -135/72 mmHg, pulse rate 48-60 times per minute, CVP 20-22 mmHg, second line arterial 66/32 mmHg to 91/36 mmHg, PAP 50/36 mmHg to 67/37 mmHg, temperature 24^{O} C – 36 C, and right brain NIRS values (51-72%), left brain (57-71%). CPB time was 193 minutes, AOX (148 minutes), ischemic time (20", 27', 21', 11'), ASCP (19 minutes), and DHCA (25 minutes).

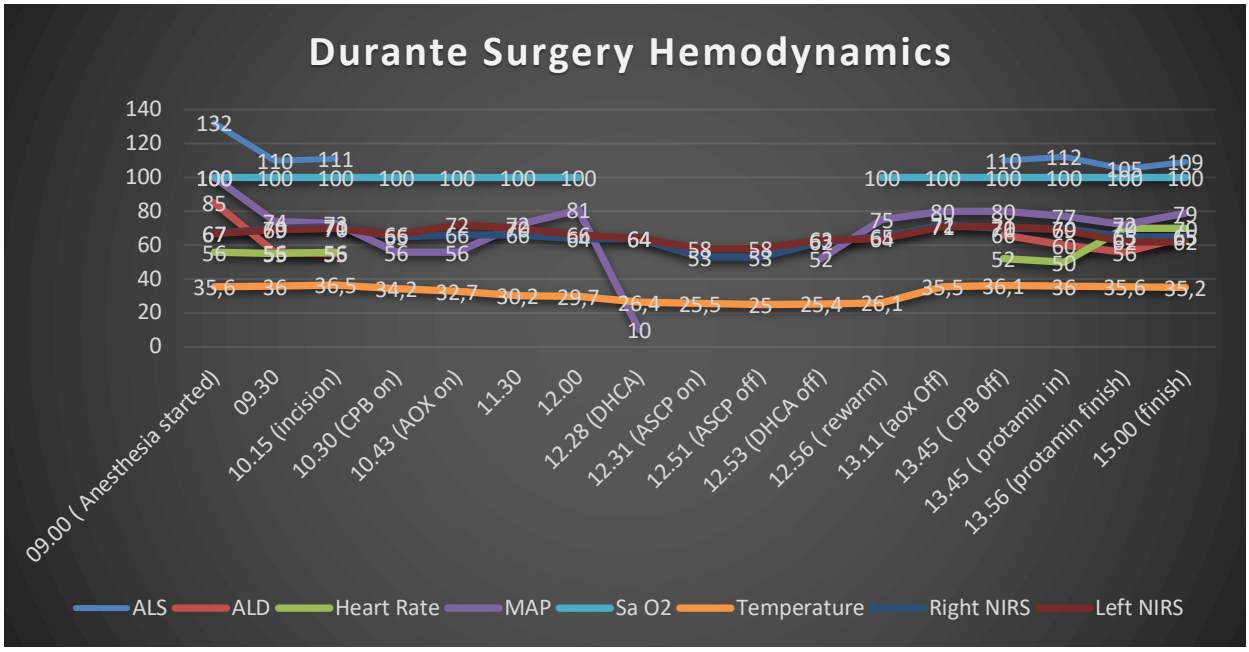

Figure 2: Durante Operation Hemodynamic diagram.

4. Postoperative Management

Postoperatively the patient was transferred to the ICU treatment room in an intubated condition and connected to a ventilator, VCV ventilator mode with 500 ml TV, RR 12 times per minute, Peep 5 mmHg, FiO2 50%, I: E = 1:2, MV 7.2 liters per minute. Hemodynamics at that time, blood pressure 128/71 mmHg, pulse rate 70 beats per minute, PAP 19/12 mmHg, CVP 9 cmH2O, temperature 35.4^{O}C, and 100% saturation with the help of dobutamine 10 mcg/kg/minute and norepinephrine 0.1 mcg /kg/minute. The patient's consciousness is still under the influence of sedative drugs, dexmedetomidine 0.2 mcg/kg/minute and the analgesic morphine 10 mcg/kg/hour. 2-hour postoperative right and substernal CTT drain about 800 cc. The patient was given transfusion therapy of 7 flasks of PRC, 8 flasks of FFP, and 3 flasks of TC. The patient was also injected with tranexamic acid 500 mg / 8 hours, vitamin K 10 mg / 8 hours, Ca gluconate 2 g and octaplex once given.

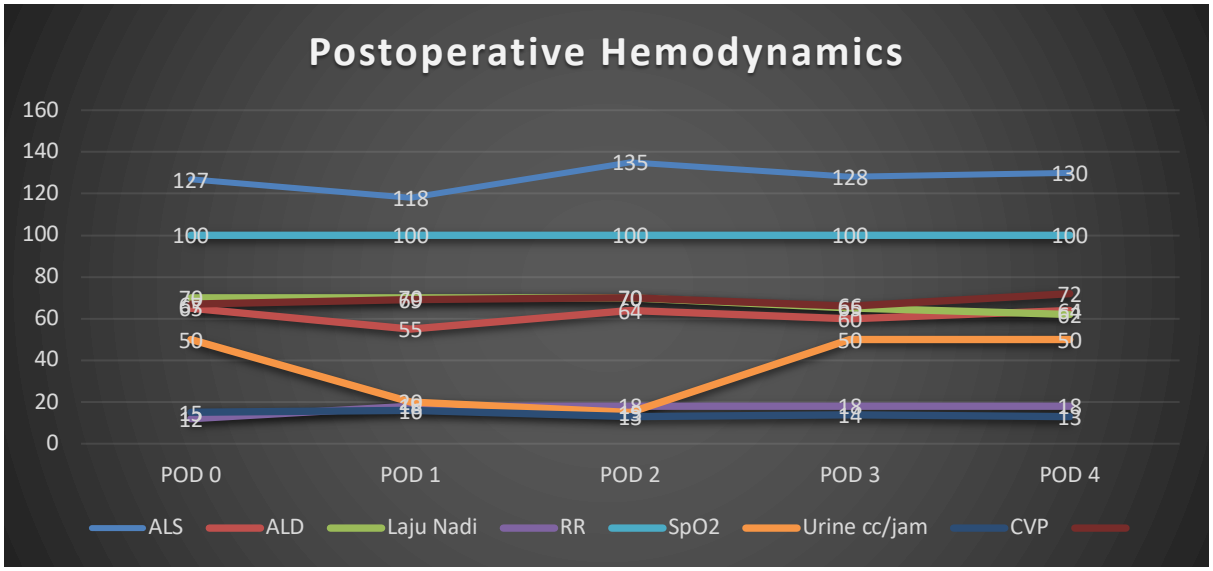

Figure 3: Postoperative Hemodynamic Diagram

The increase in postoperative kidney function was seen from laboratory examinations on the second post-op day with a creatinine value of 3.4 g/dl, an increase in potassium levels of 5.3 mmol/dl and a decrease in the amount of urine to 15-30 cc/hour. Then the patient was consulted for internal medicine to receive renal replacement therapy in the form of hemodialysis. CDL was placed in the left femoral vein followed by hemodialysis for 4 hours, urine output reached 50-100 cc/hour then the patient was extubated, and the patient moved from the ICU to the CVCU room on the fifth day.

5. Discussion

Aortic dissection is the separation of the intima and media of the aortic wall. It usually occurs as a result of a small tear in the intima (typically in the ascending aorta) where blood enters under pressure, separates the layers of the aortic wall and creates a false lumen. Propagation of the dissection is dependent on absolute blood pressure, pulse pressure and the rate of systolic arterial pressure rise (δP/δt). Distal propagation is common and, as the dissecting progresses, may occlude branches and compromise organ blood supply. Proximal extension is less common but may result in aortic valve disruption, coronary artery occlusion or rupture into the pericardium (Mackay, 2012).

The pain of aortic dissection typically is severe, abrupt in onset, and has a ripping, tearing, or stabbing quality. Highly suggestive physical findings include a pulse deficit, a systolic blood pressure differential greater than 20 mmHg, focal neurologic deficit, and a new murmur od AR. Electrocardiogram, along with urgent and definitive aortic imaging (TEE, CT, MRI), is strongly recommended in suspected aortic dissection. The most common imaging study is contras-enhanced spiral CT or CTA because it is widely available. Typical finding in acute aortic dissection includes an intimal flap, luminal displacement of intimal calcification, and aortic dilatation. Intra Mural Hematoma appears as a crescent-shaped high-attenuation thickening of aortic wall in noncontrast CT. CT can demonstrate rupture, branch-vessel involvement, and false lumen extent. Although MRI has a near 100% sensitivity and specificity and is widely available, it also takes significantly longer than CT (Kaplan, 2017).

Aortic dissection is classified according to both duration of symptoms and anatomy. Aortic dissection is termed “acute” if the diagnosis is made within 2 weeks of the initial onset of symptoms, otherwise it is termed chronic. The Stanford classification is based on involvement of the ascending aorta (type A dissection involve the ascending aorta; whereas type B does not) and is useful because of the contrasting treatment options. The DeBakey classification further subdivides dissection on an anatomic basis (Mackay, 2012). According to the above description of the signs and symptoms, physical examination of contrast CT imaging and TEE of the patient in this case was diagnosed with Stanford A Debakey Type I Aortic Dissection.

Regardless of whether acute aortic dissection is type A or B, medical therapy to control pain and hypertension is essential in all patients (Fukui, 2018). The European Cardiology Society recommends that patients with acute

aortic dissection should be managed in the intensive care setting. Morphine sulfate is recommended for pain relief. Invasive blood pressure monitoring via arterial line. Treatment is directed toward reducing aortic wall stress by reducing systolic blood pressure and the force of left ventricular ejection (Patel, 2008). Beta blockers have the desired effect of reducing blood pressure and heart rate to the normal range. These medications also protect the myocardium against ischemia. For most patients, systolic blood pressure should be controlled between 100 and 120 mmHg with a heart rate of approximately 60 bpm. Otherwise, vasodilators such as calcium channel blockers (nicardipine or diltiazem) or nitro-glycerine are useful in reducing wall stress with control of heart rate and blood pressure (Fukui, 2018). Esmolol is a particularly useful β-blocker because it has a short pharmacologic half-life and can be rapidly titrated. Esmolol can be administered as an initial bolus of 5 to 25 mg intravenously, followed by a continuous infusion of 25 to 300 μg/kg per minute. In patients with β-blocker contraindications, heart rate control should be gained with titration of nondihydropyridine calcium channel blockers such as verapamil or diltiazem. Metoprolol, a cardio selective β-blocker, may be advantageous in patients with reactive airway disease who are sensitive to β-adrenergic antagonists. Metoprolol is administered at a dose of 5 to 15 mg intravenously every 4 to 6 hours. If the systolic blood pressure remains greater than 120 mm Hg with adequate heart rate control, then vasodilators (e.g., nitroprusside at a dosage of 0.5 to 2.0 μg/kg per minute or nicardipine at a dose of 1 to 15 mg/hour) (Kaplan, 2017). In this case, the patient was given an oral -blocker along with ramipril and ivabradine (Hyperpolarization-activated Cyclic Nucleotide-gated channel blocker) which gave good results.

Table 2: Classification of Acute Aortic Dissection

DeBakey Classifications	
Type I	The entire aorta is involved (ascending, arch, and descending)
Type II	Confined to the ascending aorta
Type III	Intimal tear originating in the descending aorta with either distal or retrograde extension
Type IIIA	Intimal tear originating in the descending aorta with extension distally to the diaphragm or proximally into the aortic arch
Type IIIB	Intimal tear originating in the descending aorta with extension below the diaphragm or proximally into the aortic arch.
Stanford classifications	
Type A	Involvement of the ascending aorta and/or aortic arch regardless of the site of origin or distal extent
Type B	Confined to the descending aorta distal to the origin of the left subclavian artery

In general, the anaesthetic management of type A aortic dissection resembles the management of ascending aortic aneurysms that require DHCA. Large bore intravenous catheters are essential for intravenous medications and rapid volume expansion. A radial arterial catheter for invasive blood pressure monitoring is preferred over a femoral artery catheter to allow for CPB cannulation, depending on surgeon preference. If a pulse deficit is detected, the site for arterial pressure monitoring should be chosen to best represent the central aortic pressure. A central venous or PAC to monitor CVP, pulmonary artery pressure, and cardiac output is useful. TEE insertion is performed after anaesthetic induction, and it can be used to verify the diagnosis. The induction of general anaesthesia in hemodynamically stable patients with aortic dissection should proceed in a cautious manner. The dose of intravenous antihypertensive drugs may need to be reduced at the time of anaesthetic induction to prevent severe hypotension when combined with anaesthetic drugs. The hypertensive response to endotracheal intubation, TEE probe insertion, and sternotomy should be anticipated and attenuated with narcotic analgesics (Kaplan, 2017).

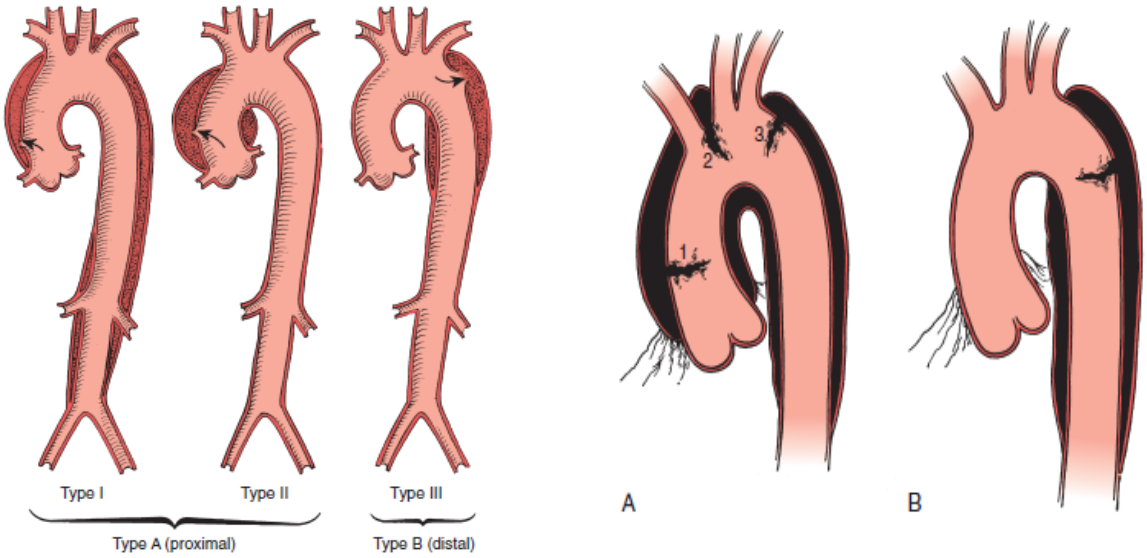

Figure 4: Debakey's classification of aortic dissection (left), Types I, II, and III. Stanford classification for aortic dissection (right) Types A and B. (from Prakash A. Patel MD, John G.T. Augustine, MD, PHASE, FAHA. Et.all, thoracic aorta: Kaplan's Cardiac Anesthesia for Cardiac and Noncardiac Surgery. 2017; 23:865)

If disease involves both the aortic valve and the aortic root, the patient requires aortic root replacement and aortic valve intervention. If technically feasible, the aortic valve can be reimplanted with a modified David technique, which includes graft reconstruction of the aortic root with reimplantation of the coronary arteries. If not feasible, aortic root replacement with a composite valve-graft conduit is indicated (Bentall procedure). Repairing aortic aneurysms that extend into or involve the aortic arch requires CPB with DHCA with or without perfusion adjuncts. For ascending aortic aneurysms that involve only the proximal aortic arch. Ascending aorta with hemiarch reconstruction often is performed using DHCA with or without ACP/RCP to make the distal anastomosis feasible without cross-clamping ("open technique"). Although the average nasopharyngeal temperature for DHCA may be about 18°C, the optimal temperature for DHCA has not been established (Kaplan, 2018). The primary method for providing protection against cerebral injury is DHCA. A period of circulatory arrest for up to 30–40 minutes at a body temperature of 15–18_C is tolerated by most patients. More recently two techniques have been developed with the aim of reducing this cerebral morbidity on the basis that some flow is better than no flow. These are retrograde cerebral perfusion (RCP) and selective anterograde cerebral perfusion (SACP). The intention of these techniques is to ensure some oxygen delivery to the brain while normal (anterograde) flow is interrupted (Mackay, 2012). The use of NIRS is a non-invasive brain monitoring to see oxygen flow, detecting the possibility of cerebral injury. After completion of the proximal anastomosis and intercostal artery-to-graft anastomoses under DHCA, the aortic graft is cannulated, and bypass flow is re-established to the upper part of the body. During a period of hypothermic low bypass flow, the distal anastomoses are completed and then rewarming is initiated (Gropper, 2020). The surgical plan for this patient was in accordance with the statement above where in the anastomose connection DHCA was performed and using SACP to deliver oxygen to the brain, NIRS was also installed for monitoring blood oxygenation to the cerebri where the decrease in the NIRS value did not exceed 20% of the basal value.

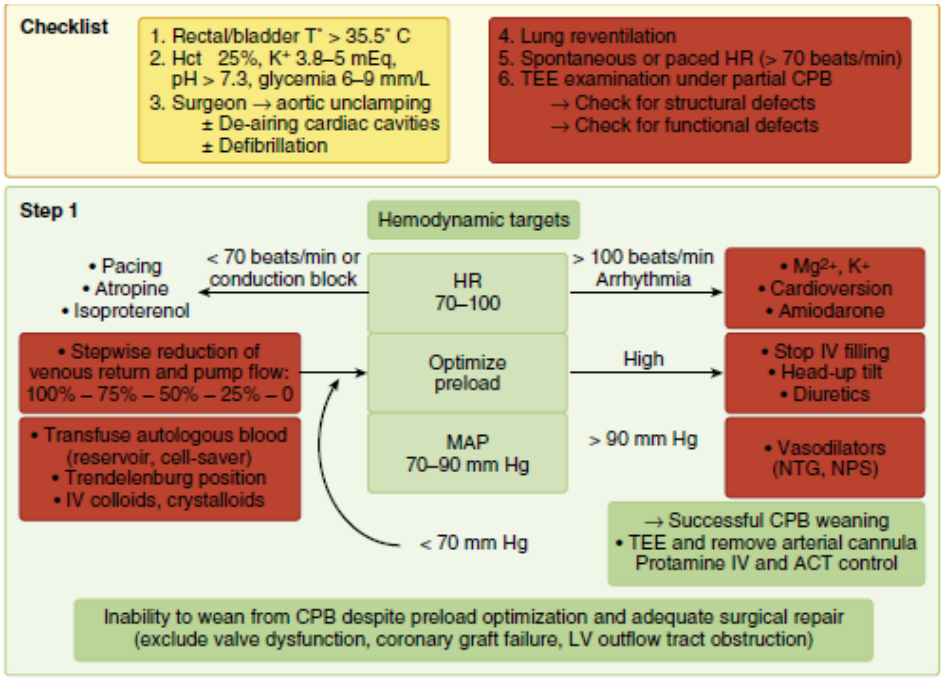

Figure 5: Cardiopulmonary bypass (CPB) release algorithm. From LickerM, Diaper J, et all. Clinical Review: management of weaning from cardiopulmonary bypass. Ann Card Anaesth, 2012; 15:206-223

Causes of death included aortic rupture, cardiac tamponade, myocardial ischemia from dissection, severe AR, stroke due to brachiocephalic dissection and malperfusion disorders including renal failure, bowel ischemia, and limb ischemia (kaplan,2017). In this case the patient had acute renal failure with a urea value of 108 and a creatinine of 2.24. In a study conducted by Hsiang in 2021 which studied the incidence of AKI in type A acute aortic dissection with a total of 696 patients, 376 patients (54%) developed AKI after surgery, from 376 patients 135 (35.9%) developed AKI. In addition, of the 320 patients without AKI, 34(10.6%) patients had worsening renal function and developed AKD. Overall, 169(24.6%) of the 696 patients had AKD after surgery for acute type A aortic dissection. Postoperatively in patients with AKI or AKD, creatinine values increased on the second and third days of treatment and decreased to baseline values on the seventh day of treatment (Hsiang, 2021). In this case, according to the theory above, where on the second day of treatment the creatinine value began to increase (Cr: 3,4 mmol/dl) and there was a decrease in the amount of urine per hour, then hemodialysis therapy was carried out for better results.

The use of an intracardiac pacemaker was carried out because after the process of releasing from the CPB machine the patient's pulse rate was bradycardia, according to the algorithm we applied for the release from the CPB machine (pictured) a pacemaker was installed, and it gave better results for the patient.

6. Conclusion

Acute aortic dissection is an emergency condition that requires immediate medical and surgical treatment. Management of pain and hypertension should be prioritized to reduce the extent of the dissection and its complications. To establish the best diagnosis with contrast and non-contrast CT can determine the classification of dissection. Anesthesia management During induction and intubation, adequate analgesia should be avoided to prevent an increase in pulse rate and blood pressure. DHCA is performed to reduce bleeding in the operative field when the anastomosis is connected with the proximal aorta. The use of SACP and NIRS for cerebral oxygenation and monitoring prevents injury to the brain. AKI is one of the complications of acute aortic dissection that often occurs and can worsen after surgery, so hemodialysis is needed to restore kidney function.

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